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CHEMICAL CONTAMINANTS, DERMAL SARCOMA AND LYMPHOCYSTIS IN SPAWNING WALLEYE FROM THE THAMES RIVER, ONTARIO

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Executive Summary

Some concern has been expressed about the role of organic contaminants in the incidence of external skin lesions on walleye from the Great Lakes. The spawning population of walleye (Stizostedion vitreum) in the Thames River, Ontario, was examined in 1987 for the incidence and histopathology of external skin lesions, and the contaminant levels in walleye (19) with conspicous lesions compared to levels in (20) visibly normal walleye. In 1987, 9% (53 of 590) of the post-spawning downstream migrants were found to exhibit skin lesions, confirmed histologically to be mostly (68%) lymphocystis with some (30%) dermal sarcoma. Muscle tissue from affected (19) fish was actually lower in polychlorinated biphenyls, hexachlorobenzene and p,p-DDE than was muscle from unaffected (20) fish. Affected walleye also had reduced muscle lipid, but the reductions in contaminants could not be explained solely by muscle lipid levels. The consumption of walleye with visible skin lesions does not increase an anglers exposure to organic contaminants in fishes flesh, compared to levels in visibly normal walleye.

Introduction

The tremendous growth (rebound) in walleye (Stizostedion vitreum Mitchell) populations throughout the Great Lakes, following reductions in commercial fishing and phosphorus discharges, has led to increased reports that many fish are diseased. Walleye and other percid fishes of North America frequently exhibit lymphocystis. This disease is manifested in cauliflower-like non-cancerous growths on the skin or fins of affected fish. These external growths are caused by an iridovirus (non-oncogenic) which infects and causes hypertrophy of connective tissue cells (Walker 1969, Roberts 1978, Colby et al. 1979, Pilcher and Fryer 1980). Dermal sarcoma, which is often found coincidently with lymphocystis, is likely caused by an oncogenic (carcinogenic) retrovirus (Walker 1969, Yamamoto et al. 1976). In this latter condition infected dermal fibroblasts proliferate to produce raised white lesions on the skin, which are often confused with lymphocystis. A third viral (retrovirus or herpesvirus) disease in walleye is epidermal hyperplasia (Walker 1969, Yamamoto et al. 1985, Kelly et al. 1983), which appears as raised pale to white lesions which are unlike lymphocystis or dermal sarcoma in appearance. The incidence of these viral diseases varies considerably throughout the year, with the highest observed rates in the spring during spawning (Ryder 1961, Rierson and Fugelli 1984, Bowser et al. 1988, D. MacLennan, unpubl. data, R. C. Haas, Department of Natural Resources, Michigan, personal communication).

Sport-fisherman and angler groups in some areas have expressed concern that walleye with lymphocystis may be unsafe to eat. While they certainly appear less palatable than unaffected walleye there are no known cases where these fish diseases have been transferred to humans. A few anglers have suggested that affected walleye may be diseased because chemical "carcinogens" have caused the "cancers" on the fishes skin, and as such anglers consuming affected walleye will ingest carcinogenic chemicals. Indeed the role of industrial contaminants in promoting the development of these viral lesions is unknown. In contrast proliferative skin lesions, which likely also involve a virus and which affect white suckers (Catostomus commersoni) and brown bullheads (Ictalurus nebulosus), are often more prevalent in polluted areas, suggesting pollution promotes their development (Baumann et al. 1987, Smith et al. 1989, Hayes et al. 1990). It is unclear if chemical pollutants affect the prevalence or severity of viral skin diseases in walleye. The evidence that chemical contaminants can cause proliferative diseases of the liver (cancer) in exposed fish populations is very good, particularly in but not limited to some populations exhibiting abnormal rates of skin lesions (Baumann et al. 1987, Smith et al. 1989, Hayes et al. 1990). Walleye populations in the Detroit River have been reported to develop liver cancer, and it has been suggested that exposure to chemical carcinogens is responsable (Kreiss et al. 1989, Benzie and Looby 1990).

The reported incidence of liver cancer in walleye from the Detroit River, and the knowledge that many Lake Erie walleye migrate through this polluted area to reach spawning areas (Thames River) has increased the concern about contaminants in these walleye among anglers in Southwestern Ontario. If exposure to chemical contaminants promoted the development of skin disorders, then affected walleye would

be more likely to come from polluted areas, and affected walleye might also be expected to have increased tissue residues of chemical contaminants. Presently the impact of these contaminants on consumption are addressed in guidelines published jointly by the Ontario Ministries of the Environment and Natural Resources (MOE 1990).

A population of walleye spawning in the Thames River, which includes walleye from the Detroit River, Lake St. Clair, the St. Clair River and lower Lake Huron (MNR 1988), was examined for evidence of lymphocystis and other viral skin disorders. Contaminant levels in visibly normal and in visibly diseased walleye were compared. Our aims were to determine if infection rates were unusually high, and if affected fish posed an increased (chemical) risk to people consuming walleye with skin lesions.

Methods

Collections

Walleye migrating downstream after spawning were collected with a 300 foot seine net at Jeanettes Creek in the lower Thames River (Figure 1) on April 22 and 23, 1987; 6 net hauls captured a total of 590 walleye. This is the same location and sampling procedure which has been used by the Ontario Ministry of Natural Resources (MNR) to assess (mark and recapture) this spawning stock (1980-82), and to provide walleye for transfers to other areas. Walleye with skin abnormalities were kept alive in tanks, and 20 walleye with more than 2 lesions or with any one lesion larger tha 1 cm. in diameter were kept for pathological examination and chemical analysis. This criteria was later adopted for definition of lesion infestations as "gross", "minor" or "absent" in a novel anglers diary program (MacLennan unpubl. data). A sample (20) of visibly normal walleye was collected whose length matched those of the affected group, to eliminate the effect of size on contaminant residues. Walleye taken to the laboratory with (20) or without (20) abnormalities were again measured for fork and total length, weighed and their sex determined. Ages were later determined by annuli on scales. All lesions were excised, fixed in 10% formalin and routinely processed through ethanols to parafin blocks for light microscopy. Sections (5 um.) stained with haemotoxylin and eosin were examined for most lesions. Skinless and boneless dorso-lateral muscle samples were taken from all 40 fish and frozen (-20°C) for chemical analysis.

Contaminant analysis

Contaminant analysis was performed by the Ontario Ministry of the Environment using procedures (MOE 1981) standard for sport-fish analysis for the development of consumption guidelines (MOE 1990). All analysis were carried out on skinless, boneless dorsal muscle fillets, which were homogenized and subsampled for the separate metals and organic determinations. Organic analysis was by capillary gas chromatography with electron capture detection. PCB, DDE, mirex, chlordanes, hexachlorobenzene and octachlorostyrene were analyzed by a single 15 m. megabore column, DB17 0.53 with a 1 um. film thickness. All other organochlorine pesticides and chlorobenzenes are run on dual columns, narrow bore DB5 and DB1701 0.25 with a 0.25 um. film thickness. Metals were analyzed by atomic absorption spectroscopy.

Statistical analysis

Because contaminant residues generally increase with fish length, the collected fish were deliberately paired by length to simplify comparisons. Paired non-parametric analysis (Mann-Whitney pairs test) was used to determine if contaminant residues differed between affected and unaffected groups. Population characteristics, growth rates (length at age) and condition factors ($(w*100)/l^3$) were tested by Students T-test and ANCOVA.

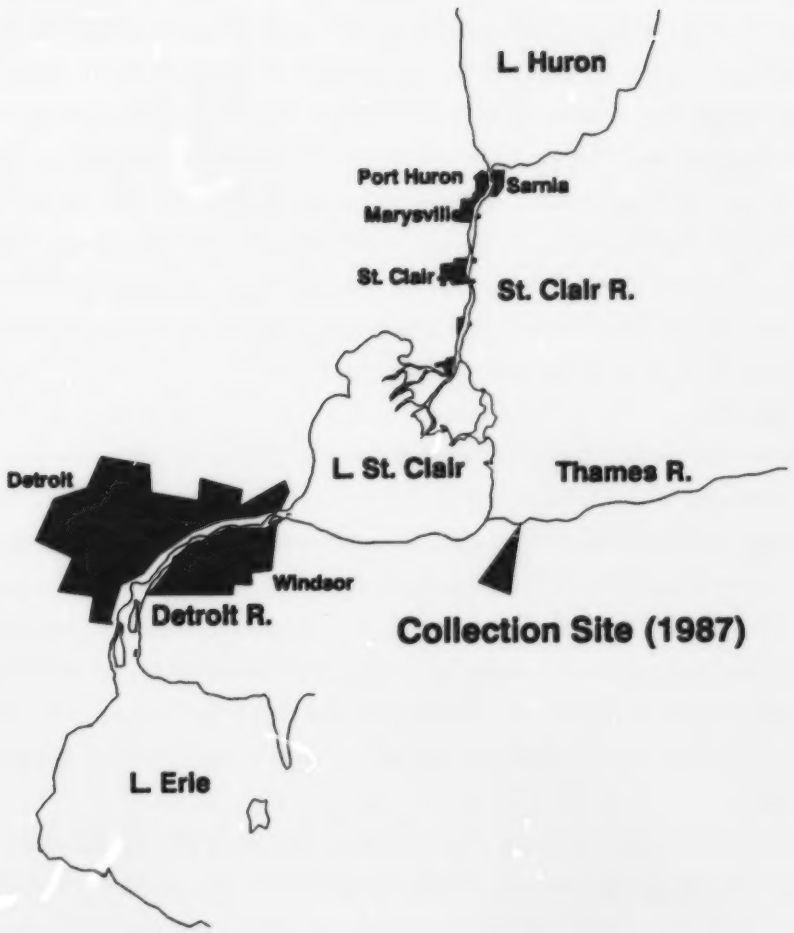

Figure 1: Map showing the collection site (Jeanettes Creek, on the Thames River). Walleye spawning in the Thames River spend the summer in L. Erie, Huron and St. Clair, as well as in the Detroit and St. Clair Rivers.

Results

Biological data

The incidence of visible skin lesions on walleye captured during the spring of 1987 was 9%, 53 of the 590 walleye captured being affected. The numbers of walleye with more than 2 lesions, or one lesion larger than 1 cm. was small (21/590, 3.6%) and 20 of these were selected for detailed studies. The sub-sample examined for pathology, contaminants and growth were clearly the most severely affected minority. The mean lengths of the affected group (43.8 cm) and a control unaffected group (43.7 cm) were similar (Table 1), as were the ages (5.63 and 5.85; not shown), suggesting these two samples were not biased by fish age or size. Growth rates (length at age) were similar (ANCOVA, $P>0.05$) in affected and unaffected walleye. In both samples the majority (19/20) of the walleye were males in post-spawning condition. Condition factors for the affected walleye (0.925) were insignificantly ($P<0.069$) lower than for the unaffected walleye (0.976).

Pathology

Histological examinations were made of 72% (79/109) of the individual lesions which affected 19 of the 20 most severely affected fish. A single walleye had 23 separate lesions covering roughly 50% of its body; these were identified as lymphocystis with a hand lens (10X), pooled, and found to include only lymphocystis. One affected fish had a single proliferative nodule subsequently identified as a mineral (calcium) deposit, commonly known as a bone spur. Lymphocystis nodules and dermal sarcomas were typical of those described in walleye (Walker 1969, Yamamoto et al 1976). Lymphocystis is comprised of extremely large cysts surronded by a hyaline sheath (Figure 2), while dermal sarcoma features homogenous masses of elongate fibrous-like cells (Figure 3), which were very discrete from the surronding dermis. The two lesions can be reliably discriminated with a 10X hand lens. Only one dermal "sarcoma" was invasive, suggesting the term dermal fibroma more accurately describes this condition; the terms dermal sarcoma or fibrosarcoma imply malignancy. Overall 68.3% of the proliferative skin lesions examined were lymphocystis, while 30.4% were dermal sarcoma, disregarding the single fish with multiple (23) lymphocystis nodules. Both conditions were found simultaneously on some fish (4/20), though some fish were affected only with lymphocystis (8/20) some only with dermal sarcoma (7/20), a single fish having the calcium deposit.

Several of the fish with grossly proliferative lesions had small hemorhagic nodules or even circular eroded (craters or sores) areas on their skin. Haemorhage and inflammation were rare in most lymphocystis nodules, but several of these sores contained remnants of lymphocystis cysts and large numbers of hemorhagic and inflammatory cells (Figure 4). It is possible that these were lymphocystis nodules pulled off during the seining, but it is more likely that they were nodules sloughed prior to capture, as evidenced by the inflammatory response prior to wound healing.

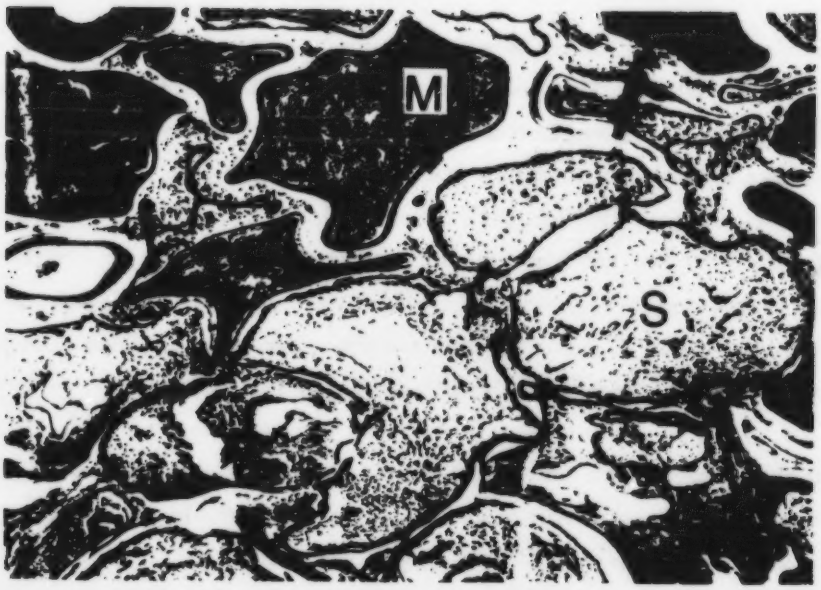

Figure 2: Photomicrograph of a lymphocystis nodule from a Thames River walleye (H and E, 1000X magnification). The large cyst-like structures are enlarged fibroblasts, either engorged with mature virus particles (M) or which have shed some virus particles (S).

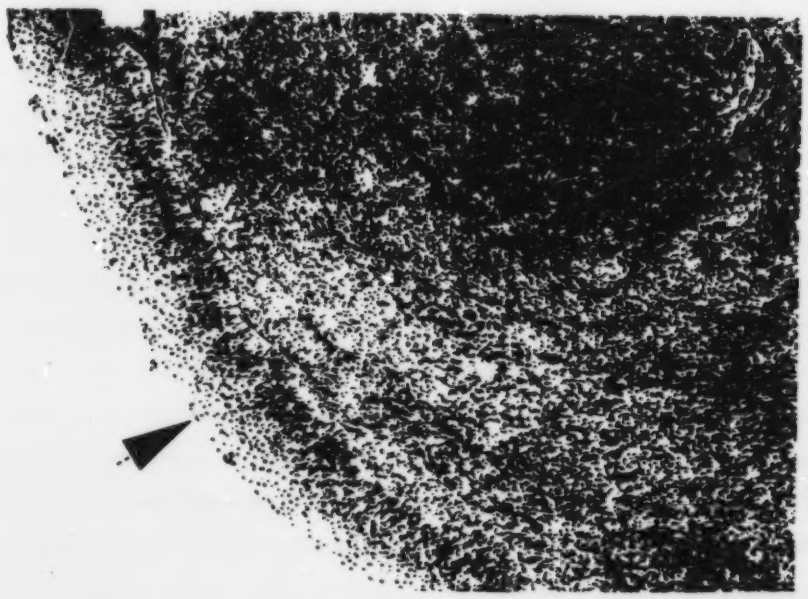

Figure 3: Photomicrograph of dermal sarcoma from a Thames River walleye (H and E, 1000X magnification). This large lesion is composed of proliferations of fibroblasts (elongate cells) interspersed with inflammatory cells, predominantly lymphocytes, below an unscaled epithelium (arrow). skin.

Contaminants

The single fish with a bone spur, rather than lymphocystis or dermal fibroma, was omitted from the following evaluation of muscle contaminants. The levels of many contaminants were below detection in more than 50% of the samples, as follows (detection limit). BHC, heptachlor and aldrin, hexachloroethane and hexachlorobutadiene, 2,4,5 and 2,3,6-trichlorotoluene, 1,2,4,5 and 1,2,3,4-tetrachlorotoluene and pentachlorobenzene (1.0 ng/gm), 1,2,3, 1,3,5 and 1,2,4-trichlorobenzene (2.0 ng/gm), mirex (5.0 ng/gm), 2,4,6 and 2,4,5-trichlorophenol, 2,3,4-trichlorophenol, 2,3,5,6 and 2,3,4,5-tetrachlorophenol and pentachlorphenol (50 ng/gm), nickel, manganese, cadmium and chromium (0.4 ug/gm) were below detection. Residues of alpha-chlordane were detected in > 50% but < 100% of the samples, so the detection limit was used in subsequent calculations for samples below detection. The levels of the remainder of the contaminants (Table 1) were above detection in all samples. Lipid, PCB and p,p-DDE residues were significantly lower ($P<0.03$) in the sub-sample with skin lesions than in the sub-sample of normal walleye, though lead residues were higher ($P<0.004$) in the affected sub-sample (Table 1). Correction of the PCB and p,p-DDE residues for the differences in lipid reduced the differences somewhat between groups, however PCB and p,p-DDE levels were still significantly lower ($P<0.02$) in the affected walleye (Table 1).

An abundance of historical data was also available for this region which has been gathered for the publication of consumption guidelines (MOE 1990). Because of the overall decline (visible in PCB and mercury levels for several samples in Table 2) in most contaminants in the Great Lakes over time, only data from 1985 to 1989 was considered. Mercury levels in walleye taken for the present study are similar to historical levels in Lake St. Clair and the St. Clair River, which are clearly higher than found downstream in the Detroit River or L. Erie (Table 2). In contrast the levels of PCBs in the present study are much higher than from most areas. This may be because only males were analyzed; females tend to have less PCB because of excretion of lipid soluble chemicals in the eggs. The present levels most closely resemble an old (1985) collection of walleye from the St. Clair River (Table 2), clearly exceeding older (1986) levels in other areas. A plot of PCB residues in individual fish (Figure 5) shows that 3 normal and 3 diseased walleye had abnormally high PCB levels, >600 ng/gm, which are remarkably similar to PCB levels seen in larger specimens from this 1985 sample of walleye from the St. Clair River. The observation that 6/39 (15%) had PCB and mercury residues typical of the St. Clair R. walleye reflects their distribution based on tag recovery data which showed 30% of the walleye tagged in the Thames R. (1980-82) were subsequently caught in the St. Clair River (MNR 1988). The remainder of the present sample had PCB and mercury residues typical of Lake St. Clair, which accounted for 41% of the recoveries of walleye tagged (1980-82) in the Thames River (MNR 1988).

Table 1: Contaminant residues and biological data for normal walleye and walleye with proliferative skin disorders captured from the Thames River in 1987.

		Normal Walleye (N=20)			Diseased Walleye(N=19)			
Parameter*	Detection	Mean	S.D.	Range	Mean	S.D.	Range	Significance
Length	-	46.5	4.69	37.5-55.2	46.3	4.49	37.5-55.9	NS
Weight	-	831	251	440-1370	788	240	470-1410	NS
Mercury	0.03	0.45	0.19	0.16-0.96	0.43	0.20	0.12-0.92	NS
Copper	0.03	0.39	.012	0.28-0.71	0.31	0.20	0.19-1.1	NS
Lead	0.01	0.08	0.03	0.008-0.14	0.098	0.2	0.018-0.92	.004
Zinc	0.03	2.44	0.84	1.5-5.1	2.02	0.96	0.81-3.6	NS
Arsenic	0.03	0.096	0.058	0.003-0.24	0.058	0.021	0.03-0.1	NS
Selenium	0.03	0.53	0.11	0.36-0.79	0.63	0.28	0.27-1.6	NS
PCB	20	294	216	82-755	189	239	20-785	.003
Hexachlorobenzene	1.0	2.2	1.15	1.0-6.0	1.05	0.22	1-2	.0001
p,p-DDT	2	4.95	3.9	2-13	2.6	2.8	2-14	NS
p,p-DDD	2	2.85	3.8	2-19	2.5	1.6	2-7	NS
p,p-DDE	5.0	54.3	53.2	10-215	16.1	35.4	1-158	.0001
Chlordane	2.0	2.8	1.4	2-6	3.2	2.5	2-10	NS
Octachlorostyrene	1.0	5	3.8	1-13	2.9	1.9	1-6	NS
lipid (%)	-	1.71	0.42	1.12-2.48	1.43	0.42	0.88-2.72	.03
PCB lipid corrected	-	163	97	60-422	116	118	21-438	.02
p,p-DDE/lipid	-	29.7	26.5	7-96	8.5	13.4	0.7-58	.000006

* Units are cm. for length, gm. for weight, ug/gm for metals, ng/gm for organics and percent (%) for lipids, all based on wet weight

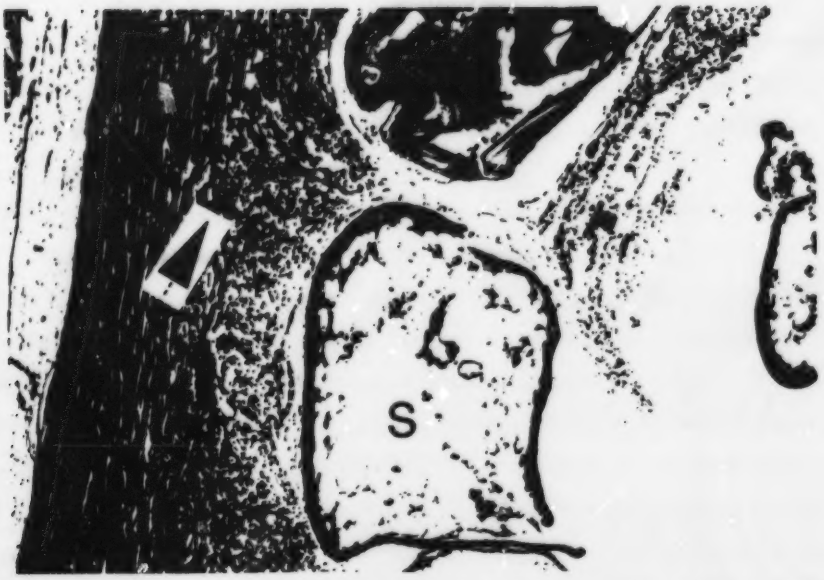

Figure 4: Photomicrograph of a red sore from a male Thames River walleye (H and E, 2000X magnification). The rupture of this lesion has exposed the dermis (arrow) to the water, resulting in a pronounce inflammatory reaction. The partially collapsed lymphocystis cysts which has shed its mature viruses (S) clearly contrasts with a mature cyst still retaining virus particles. Such an exposed area of skin is a likely avenue for infection by other disease organisms.

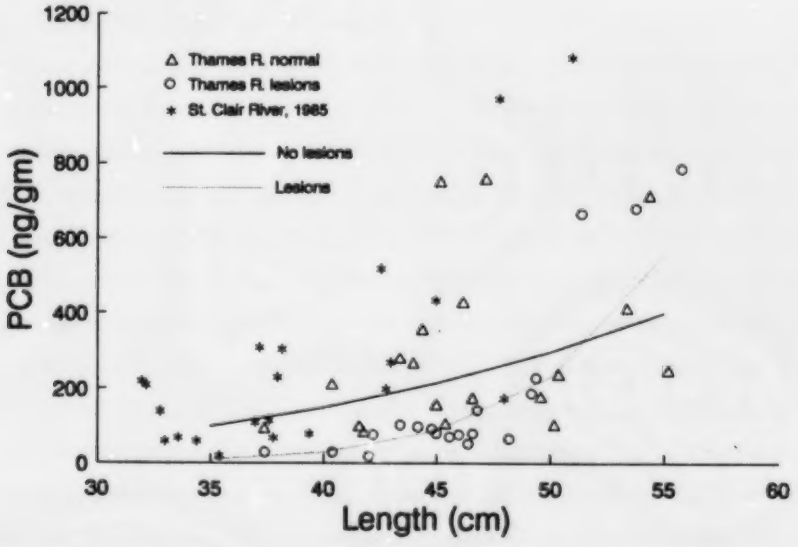

Figure 5: PCB residues in walleye from the present study (normal and lesions) and a 1985 collection from the St. Clair River. The lower PCB residues in walleye with lesions (than normal walleye) is shown as the non-linear least squares estimate (regression) for each group. The presence of 6 walleye with unusually high PCB residues is also apparent, only approaching those of several walleye from the 1985 St. Clair collection.

Table 2: Historical and current data for mercury and PCB in walleye populations in Southwestern Ontario.

Location	Condition	Date, Season	N	Length (cm)	Weight (gm)	Mercury (ug/g)	PCB (ng/g)
Thames River	Normal	1987, Spring	20	46.5	831	0.45	294
	Diseased	1987, Spring	19	46.3	788	0.43	189
	Normal	1989, Spring	30	47.3	954	0.36	133
L. St. Clair	Normal	1986, Fall	23	46.8	1024	0.43	118
	Normal	1988, Fall	30	45.6	866	0.37	NA
St. Clair R.	Normal	1985, Summer	20	38.5	606	0.35	273
Detroit River	Normal	1986, Fall	19	39.9	593	0.20	162
	Normal	1988, Fall	20	42.8	940	0.18	43
W. Lake Erie	Normal	1986, Fall	20	39.9	593	0.20	208
	Normal	1988, Fall	20	52.7	1544	0.14	46

Discussion

The incidence of lymphocystis and dermal fibroma was 9% (53 of 590) in the sample of predominantly post-spawning male walleye taken for this study. Contaminant levels were actually reduced in walleye with skin lesions, compared to visibly normal walleye, though reduced condition factors and lipid levels were evident in walleye with skin lesions.

In three previous years (1980-82) during the upstream (pre-spawning) migration incidences ranged from 3.2 to 5.7% (N > 6000 per year) suggesting the subsamples in the present study were fairly typical of this population in the spring (D. MacLennan, unpubl. data), given that the focus of the 1987 collections was to capture walleye with lesions, while the earlier focus was to tag large numbers of walleye. Seasonal variations in lesion prevalence, peaking at spawning and lowest in the late summer (Ryder 1961, Rierson and Fugelli 1984, Bowser et al 1988) require comparisons between studies be made with regard to the season. Studies of the western Lake Erie sub-population (R.C. Haas, Michigan Department of Natural Resources, pers. comm.) found infection rates of 7-8% in the spring and 4-6% in the summer. Similarly Kreiss et al. (1989) found 4.5% of the walleye (N=66) in the polluted Detroit River to be affected in the summer. Margenau et al. (1988) found 18% of the walleye from the polluted St. Louis River to be affected in the spring. These rates, for the oft-stated "polluted Great Lakes" compare with infection rates (spawning) in pristine areas of Ontario of 25-29% (Ryder 1961), 11% and 8% in Saskatchewan (Yamamoto et al. 1976, 1985), and 18% in Lake Oneida (Bowser et al. 1988). The higher rate of infection in inland populations unaffected by significant pollution clearly refutes any suggestion that the rates in the Thames River population are abnormally high because of pollution.

The present study found no significant differences in growth rates (length at age, ANCOVA, $P < 0.725$) between affected and unaffected walleye, albiet in a very small sample, but the near significant reduction in condition factors (Students T-test, $p<0.069$) suggests additional studies of minor health effects are warranted. Analysis of data obtained in a more detailed study of skin lesions in 1989 (Thames R.) found that condition factors were not different, but growth rates (length at age) were reduced in affected walleye of both sexes (I. Smith, unpublished data). The lesions themselves may not be lethal or affect fish growth significantly, perhaps because the rapid decline in prevalence noted by some authors, due to sloughing of the lesions (Roberts 1978, Colby et al. 1979), limits their effects. This sloughing may however produce a wound which could become infected with other diseases. The high prevalence (up to 30%) of proliferative skin disorders in many walleye populations examined at spawning time (Ryder 1961, Yamamoto et al. 1976, Colby 1979, Margenau et al. 1988, Bowser et al. 1988) means that any impact of the disease could markedly affect walleye populations. It is not clear if conditions such as the four skin disorders common on North American walleye impact on the "health" of affected fish. One report, based on a mark and recapture program, suggests increased mortality for affected fish (Margenau et al 1988), but this is refuted by others (Ryder 1961, R.C. Haas Michigan Department of Natural Resources, personal communication). These studies have

failed to consider the regression of lesions in calculating survival data from returns. More subtle effects noted include reduced weight at known age (Hiles 1954) in Saginaw Bay walleye, although data from Lake Erie sub-populations suggests no impact (R. Haas, pers. comm.). Large scale effects studies (for example McFarlane and Beamish 1990) which show reduced growth in tagged fish suggest external abnormalities can produce a measurable impact, but such studies in walleye must address lesion regression.

The finding that the skin lesions on Thames River walleye were predominantly lymphocystis (68%) but with some dermal sarcoma (30%) as well is a marked contrast to previous studies. Yamamoto et al. (1976) and Bowser et al. (1988) found the converse, 66-77% dermal sarcoma and 23-33% lymphocystis. Another disease complex characteristic of wallye is "discrete" and "diffuse" epidermal hyperplasia, first noted by Walker (1969) and subsequently detailed by Yamamoto et al. (1985) and Kelly et al. (1983). These diseases, caused by retro-viruses and herpesvirus respectively, also exhibit seasonal trends in prevalence (Yamamoto et al. 1985, Bowser et al. 1988). This complex was not noted in the present study, likely because the walleye collected for examination were the most severly affected. Epidermal hyperplasia is a lesion easily missed by fisheries crews during tagging or large-scale operations, as well as by fisherman, although subsequent investigations (1989) have found this condition on 15.2% (N=203) of the Thames River walleye (I.R. Smith, unpublished data). Field studies that include a high degree of vigilance to specifically detect all forms of skin lesions yield a higher incidence, as noted in 1989 (44.1%, N=203) in the Thames River (I.R. Smith, unpublished data). While this is higher than that reported (10-22%) in central Saskatchewan Lakes (Yamamoto et al. 1985) and (32%) in Lake Oneida (Bowser et al. 1988) when all forms of skin lesions were enumerated, it does not necessarily hold that pollution affecting the Thames River walleye has resulted in increased incidences of hyperplasia.

One type of lesion identified in the Thames River walleye which was unusual were the "red sores" found on several fish, which contained defunct lymphocystis cysts. Future investigations of post-spawning or summer-time walleye must also address the incidence (4.9% in 1989 collections) of "red sores" as the presence of defunct lymphocystis capsules in these lesions suggests they are part of the disease complex, possibly lesions which have sloughed off or are regressing after spawning. Such lesions may be effective portals for the entry of virulent pathogens which could infect and cause diseases in post-spawning walleye.

The failure of affected walleye to have higher levels of contaminants than did unaffected fish suggests that contaminant levels do not correlate with any likelihood to develop skin lesions, further suggesting pollution does not affect the incidence of this disease. This is a marked contrast from the incidence of epidermal papillomas (likely viral) in bullheads and suckers, which is present in 15-35% of the population in clean areas, but affects 55-60% of the fish in polluted areas (Smith et al. 1979, Hayes et al. 1990). The lower levels of some contaminants found in walleye with skin lesions compared to normal walleye may be coincidental, or may themselves be indicative of a sub-lethal effect of the lesions. Perhaps walleye with skin lesions become infected because of reduced nutrition, due to differences in prey availability

or suitability, feeding preferences or habitat differences? Possibly males with lesions are less suitable or successful at spawning, and stay on the spawning beds longer than unaffected fish, burning up energy stores (lipid) and associated contaminants in the process. However, reduced muscle contaminants, lipids and condition factors could all be accounted for by a relatively recent reduction in predatory efficiency (consumption of fewer contaminated prey organisms) and in swimming performance. Increased hydraulic drag due to proliferations on the skin would require more muscle lipid be used for energy during the spawning (upstream) migrations, but drag induced lipid mobilization might not account for the reduced levels of contaminants after correction for tissue lipids. This latter difference is best explained by a reduction in efficiency at catching lipid-rich (contaminated) prey.

Clearly the presence of proliferative skin lesions does not represent a "marker" for contaminated walleye, but evidence for reduced lipids, contaminants and condition factors in affected walleye suggests that this disease complex may itself impact on the health of affected fish.

Conclusions

1) The incidence of external lesions in walleye from the spawning migration, 9%, is not unusually high compared to other walleye populations, suggesting contaminants in Lakes St. Clair, Erie and Huron, and the St. Clair and Detroit Rivers, do not induce an abnormal incidence of these lesions.

2) The external lesions on these walleye were lymphocystis and dermal sarcoma, both of which are caused by viruses.

3) Contaminant levels in walleye with skin lesions were lower than in visibly normal walleye, suggesting that no unusual risk (contaminant) is associated with their consumption by anglers.

4) Reduced muscle lipid, contaminant levels (lipid corrected) and condition factors may be suggestive of a sublethal impact on the health of affected fish.

Recommendations

1) Guidelines for the consumption of walleye should not be altered in any way to consider walleye with visible skin lesion.

2) A note should be added to the "Guide To Eating Ontario Sport Fish" indicating the results of this study.

3) Future investigations by Ontario Ministry of Natural Resources personell should address the incidence of lymphocystis, dermal sarcoma, discrete and diffuse epidermal hyperplasia in walleye, for any evidence that the incidence of the latter two conditions is altered by exposure to chemical contaminants.

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